AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph that appears at page 1, line 15 through page 2, line 23, with the following amended paragraph:

A major histopathological hallmark of Alzheimer's Disease (AD) is the presence of amyloid deposits within neuritic and diffuse plaques in the parenchyma of the amygdala, hippocampus and neocortex (Glenner and Wong, 1984; Masters et al., 1985; Sisodia and Price, 1995). Amyloid is a generic term that describes fibrillar aggregates that have a common β-pleated structure. These aggregates exhibit birefringent properties in the presence of Congo red and polarized light (Glenner and Wong, 1984). The diffuse plaque is thought to be relatively benign in contrast to the neuritic plaque which appears to be strongly correlated with reactive and degenerative processes (~~Dickson~~ Dickerson et al., 1988; Tagliavini et al., 1988; Yamaguchi et al., 1989; Yamaguchi et al., 1992). The principal component of neuritic plaques is a 42 amino acid residue amyloid-β (Aβ) peptide (Miller et al., 1993; Roher et al., 1993) that is derived from the much larger β-amyloid precursor protein, βAPP (or APP) (Kang et al., 1987). Two major C-terminal variants of amyloid-β peptide, Aβ 1-40 ending at Val40 and Aβ 1-42(43) ending at Ala42 or Thr43, proteolytically cleaved from βAPP, were found in amyloid deposits (Miller et al., 1993; Roher et al., 1993). Aβ 1-42 is produced less abundantly than the 1-40 Aβ peptide (Haass et al., 1992; Seubert et al., 1992), but the preferential deposition of Aβ1-42 results from the fact that this COOH-extended form is more insoluble than 1-40 Aβ and is more prone to aggregate and form anti-parallel β-pleated sheets (Joachim et al., 1989; Halverson et al., 1990; Barrow et al., 1992; Burdick et al., 1992; Fabian et al., 1994). Aβ 1-42 can seed the aggregation of Aβ 1-40 (Jarrett and Lansbury 1993). Iwatsubo et al., (1996) and Saido et al., (1996) further reported that other variant amyloid-β peptides, Aβ 3(pyroglutamate)-42, Aβ 11(pyroglutamate)-42, Aβ 17-42, Aβ 1 (D-Asp)-42, and Aβ 1 (L-isoAsp)-42 were also found to be present in amyloid deposits in the brain.

Please replace the paragraph that appears at page 6, line 16 through page 7, line 9, with the following amended paragraph:

Given that neurotoxicity appears to be associated with β-pleated aggregates of Aβ, one therapeutic approach is to inhibit or retard Aβ aggregation. The advantage of this approach is that the intracellular mechanisms triggering the overproduction of Aβ or the effects induced intracellularly by Aβ need not be well understood. Various agents that bind to Aβ are capable of inhibiting Aβ neurotoxicity in vitro, for example, the Aβ-binding dye, Congo Red, completely inhibits Aβ-induced toxicity in cultured neurons (Yankner et al., 1995). Similarly, the antibiotic ~~rifampacin~~ rifampicin also prevents Aβ aggregation and subsequent neurotoxicity (Tomiyama et al., 1994). Other compounds are under development as inhibitors of this process either by binding Aβ directly, e.g., hexadecyl-N-methylpiperidinium (HMP) bromide (Wood et al., 1996), or by preventing the interaction of Aβ with other molecules that contribute to the formation of Aβ deposition. An example of such a molecule is heparan sulfate or the heparan sulfate proteoglycan, perlecan, which has been identified in all amyloids and is implicated in the earliest stages of inflammation associated amyloid induction.

Please replace the last line on page 43 with the following amended line:

Sisodia et al., *FASEB* 9:366-370[[369]] (1995).